WEST

Freeform Search

Database: US Patents Full Text Database
US Pre-Grant Publication Full Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Term:

Display: 10 **Documents in Display Format:** CIT **Starting with Number** 1

Generate: ○ **Hit List** ◉ **Hit Count** ○ **Image**

Search | Clear | Help | Logout | Interrupt

Main Menu | Show S Numbers | Edit S Numbers | Preferences

Search History

T day's Date: 8/7/2001

DB Name	Query	Hit Count	Set Name
USPT,PGPB,DWPI	(screen or screening) same (I19)	3	L21
USPT,PGPB,DWPI	I1 and I19	11	L20
USPT,PGPB,DWPI	(ar nox) or (ar-nox) or (nadh oxidase)	141	L19
USPT,PGPB,DWPI	I17 and I5	2	L18
USPT,PGPB,DWPI	(screen or screening) same (I2)	89	L17
USPT,PGPB,DWPI	I15 not I10	4	L16
USPT,PGPB,DWPI	I1 and I5	9	L15
USPT,PGPB,DWPI	(I13) not (I10 or I11)	22	L14
USPT,PGPB,DWPI	I1 and I2	22	L13
USPT,PGPB,DWPI	9515812.pn.	2	L12
USPT,PGPB,DWPI	I2 and I5 and I6	4	L11
USPT,PGPB,DWPI	I1 and I5 and I6	5	L10
USPT,PGPB,DWPI	I1 and I2	22	L9
USPT,PGPB,DWPI	spectrophotometric	9362	L8
USPT,PGPB,DWPI	superoxide dimutase or sod or sodm	3724	L7
USPT,PGPB,DWPI	cytochrome c or cyt c	2489	L6
USPT,PGPB,DWPI	(ubiquinone) or (co q) or (coenzyme q)	2037	L5
USPT,PGPB,DWPI	(dye or enzyme or isotope or fluorescent or luminescent)	465553	L4
USPT,PGPB,DWPI	(label or labeled) same (dye or enzyme or isotope or fluorescent or luminescent)	24088	L3
USPT,PGPB,DWPI	(ar nox) or (nox) or (nadh oxidase)	24892	L2
USPT,PGPB,DWPI	((435/4)!.CCLS.)	2256	L1

Trying 3106016892...Open

Welcome to STN International! Enter x:x
LOGINID:ssspta1619lxw
PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Dec 17	The CA Lexicon available in the CAPLUS and CA files
NEWS 3	Feb 06	Engineering Information Encompass files have new names
NEWS 4	Feb 16	TOXLINE no longer being updated
NEWS 5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS 6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS 7	May 07	DGENE Reload
NEWS 8	Jun 20	Published patent applications (A1) are now in USPATFULL
NEWS 9	JUL 13	New SDI alert frequency now available in Derwent's DWPI and DPCI

NEWS EXPRESS	July 11 CURRENT WINDOWS VERSION IS V6.0b, CURRENT MACINTOSH VERSION IS V5.0C (ENG) AND V5.0JB (JP), AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2001
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 12:55:54 ON 07 AUG 2001

=> fil caplus uspatfull embase biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

FILE 'CAPLUS' ENTERED AT 12:56:09 ON 07 AUG 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE 'USPATFULL' ENTERED AT 12:56:09 ON 07 AUG 2001

FILE 'EMBASE' ENTERED AT 12:56:09 ON 07 AUG 2001
COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 12:56:09 ON 07 AUG 2001
COPYRIGHT (C) 2001 BIOSIS(R)

=> s (ar-nox) or (ar nox) or (nadh oxidase)

L1 4194 (AR-NOX) OR (AR NOX) OR (NADH OXIDASE)

=> dup rem l1

PROCESSING IS APPROXIMATELY 29% COMPLETE FOR L1
PROCESSING IS APPROXIMATELY 63% COMPLETE FOR L1
PROCESSING IS APPROXIMATELY 93% COMPLETE FOR L1
PROCESSING COMPLETED FOR L1
L2 2559 DUP REM L1 (1635 DUPLICATES REMOVED)

=> s ubiquinone or coenzyme q or co q

L3 17262 UBIQUINONE OR COENZYME Q OR CO Q

=> s cytochrome c

L4 78254 CYTOCHROME C

=> s ascorbate

L5 49891 ASCORBATE

=> d his

(FILE 'HOME' ENTERED AT 12:55:54 ON 07 AUG 2001)

FILE 'CAPLUS, USPATFULL, EMBASE, BIOSIS' ENTERED AT 12:56:09 ON 07 AUG 2001
L1 4194 S (AR-NOX) OR (AR NOX) OR (NADH OXIDASE)
L2 2559 DUP REM L1 (1635 DUPLICATES REMOVED)
L3 17262 S UBIQUINONE OR COENZYME Q OR CO Q
L4 78254 S CYTOCHROME C
L5 49891 S ASCORBATE

=> s l2 and l3 and l4 and l5

L6 9 L2 AND L3 AND L4 AND L5

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7 9 DUP REM L6 (0 DUPLICATES REMOVED)

=> d ibib abs

L7 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:706975 CAPLUS
DOCUMENT NUMBER: 133:276372
TITLE: Methods for identifying agents that inhibit serum aging factors (**NADH oxidase**) and uses and compositions thereof
INVENTOR(S): Morre, Dorothy M.; Morre, D. James
PATENT ASSIGNEE(S): Purdue Research Foundation, USA
SOURCE: PCT Int. Appl., 43 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000057871	A2	20001005	WO 2000-US8433	20000329

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-126894 P 19990330

AB The invention described here relates to methods for prevention or treatment of disorders caused by oxidative damage resulting from generation of reactive oxygen species by an aging-specific isoform of **NADH oxidase** (**AR-NOX**). The invention encompasses methods of assaying, screening, and identifying agents that inhibit **AR-NOX**, as well as methods using **ubiquinone** to inhibit the ability of **AR-NOX** to generate reactive oxygen species. These agents may be formulated into pharmaceutical compns. in the prevention and treatment of disorders caused by oxidative damage, such as cancer, diabetes, parkinsonism, atherosclerosis, cardiotoxicity, nephrotoxicity, autoimmune diseases, etc.

=> d 2 ibib abs

L7 ANSWER 2 OF 9 USPATFULL
ACCESSION NUMBER: 1998:150713 USPATFULL
TITLE: Bioassay for toxic substances activated by metabolic enzyme system
INVENTOR(S): Read, Harry W., Madison, WI, United States
Gustavson, Karl, Madison, WI, United States
Blondin, George A., Madison, WI, United States
PATENT ASSIGNEE(S): Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5843696		19981201	
APPLICATION INFO.:	US 1995-551384		19951101	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Knight, John
ASSISTANT EXAMINER: Jones, Dameron
LEGAL REPRESENTATIVE: Quarles & Brady
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 878
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method for bioassaying for metabolic activation of toxicants from a xenobiotic compound by a metabolic enzyme system includes incubating the xenobiotic compound with a metabolic enzyme system known to produce

toxicants during normal metabolic degradation processes and with a mitochondrial membrane preparation competent for enzymatic electron transfer. The production of a toxicant has a detrimental effect upon the electron transfer activity of the mitochondrial membrane preparation which can readily be assayed by observing changes in concentration of a selected redox indicator.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d 3 ibib abs

L7 ANSWER 3 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:82388 BIOSIS
DOCUMENT NUMBER: PREV199799374101
TITLE: Lipid peroxidation and changes in the **ubiquinone** content and the respiratory chain enzymes of submitochondrial particles.
AUTHOR(S): Forsmark-Andree, Patrik (1); Lee, C.-P.; Dallner, Gustav; Ernster, Lars
CORPORATE SOURCE: (1) Dep. Biochem., Arrhenius Lab., Natural Sci., Univ. Stockhom, S-106 91 Stockholm Sweden
SOURCE: Free Radical Biology & Medicine, (1997) Vol. 22, No. 3, pp. 391-400.
ISSN: 0891-5849.
DOCUMENT TYPE: Article
LANGUAGE: English

AB The relationship between lipid peroxidation induced by **ascorbate** and adenosine ADP/Fe-3+, and its effect on the respiratory chain activities of beef heart submitochondrial particles has been investigated.

Lipid peroxidation, measured as thiobarbituric acid reactive substance formation, resulted in an inhibition of the NADH and succinate oxidase activities. Examination of several partial reactions of the respiratory chain revealed inactivation primarily of those involving endogenous **ubiquinone**, i.e., NADH- and succinate-**ubiquinone**, and **cytochrome c** reductases. Ubiquinol-**cytochrome c** reductase, measured with reduced **ubiquinone**-2 as electron donor, was unaffected. The amount of NADH- or succinate-reducible cytochrome b in the presence of cyanide was strongly decreased, but could be recovered by the addition of antimycin. There occurred a substantial decrease of the **ubiquinone** content in the course of lipid peroxidation, with a linear relationship between this decrease and the NADH and succinate oxidase activities. The results are consistent with the conclusion that the **ubiquinone** pool undergoes an oxidative modification during lipid peroxidation, to a form that can no longer function as a component of the respiratory chain. Lipid peroxidation also led to a partial inhibition of the succinate dehydrogenase and **cytochrome c** oxidase activities and a minor decrease of the **cytochrome c** and cytochrome a contents. Reduction of endogenous **ubiquinone** prevented lipid peroxidation as well as the concomitant modification of **ubiquinone** and inactivation of the respiratory chain. These observations suggest that the destruction of **ubiquinone** through lipid peroxidation is the primary cause of inactivation of the respiratory chain, and emphasize the antioxidant role of ubiquinol in preventing these effects. The possible implications of these findings for regulation of the cellular turnover of **ubiquinone** by the prevailing oxidative stress are discussed.

=> d 4 ibib abs

L7 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:21582 CAPLUS
DOCUMENT NUMBER: 126:102083
TITLE: Lipid peroxidation and changes in the **ubiquinone** content and the respiratory chain enzymes of submitochondrial particles
AUTHOR(S): Forsmark-Andree, Patrik; Lee, C.-P.; Dallner, Gustav; Ernster, Lars
CORPORATE SOURCE: Div. Medical Cell Biology, Karolinska Inst., Huddinge, S-141 86, Swed.
SOURCE: Free Radical Biol. Med. (1996), Volume Date 1997, 22(3), 391-400
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The relationship between lipid peroxidn. induced by **ascorbate** and adenosine ADP/Fe3+, and its effect on the respiratory chain activities of beef heart submitochondrial particles has been investigated. Lipid peroxidn., measured as thiobarbituric acid reactive substance formation, resulted in an inhibition of the NADH and succinate oxidase activities. Examn. of several partial reactions of the respiratory chain revealed inactivation primarily of those involving endogenous **ubiquinone**, i.e., NADH- and succinate-ubiquinone1 and **cytochrome c** reductases. Ubiquinol-**cytochrome c** reductase, measured with reduced ubiquinone2 as electron donor, was unaffected. The amt. of NADH- or succinate-reducible cytochrome b in the presence of cyanide was strongly decreased, but could be recovered by the addn. of antimycin. There occurred a substantial decrease of the **ubiquinone** content in the course of lipid peroxidn., with a linear relationship between this decrease and the NADH and succinate oxidase activities. The results are consistent with the conclusion that the **ubiquinone** pool undergoes an oxidative modification during lipid peroxidn., to a form that can no longer function as a component of the respiratory chain. Lipid peroxidn. also led to a partial inhibition of the succinate dehydrogenase and **cytochrome c** oxidase activities and a minor decrease of the **cytochrome c** and cytochrome a contents. Redn. of endogenous **ubiquinone** prevented lipid peroxidn. as well as the concomitant modification of **ubiquinone** and inactivation of the respiratory chain. These observations suggest that the destruction of **ubiquinone** through lipid peroxidn. is the primary cause of inactivation of the respiratory chain, and emphasize the antioxidant role of ubiquinol in preventing these effects. The possible implications of these findings for regulation of the cellular turnover of **ubiquinone** by the prevailing oxidative stress are discussed.

=> d 5 ibib abs

L7 ANSWER 5 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1996:321743 BIOSIS
DOCUMENT NUMBER: PREV199699044099
TITLE: Mode of antibacterial action of totarol, a diterpene from Podocarpus nagi.
AUTHOR(S): Haraguchi, Hiroyuki (1); Oike, Shingo; Muroi, Hisashi; Kubo, Isao
CORPORATE SOURCE: (1) Fac. Eng., Fukuyama Univ., Gakuen-cho, Fukuyama 729

Japan
SOURCE: Planta Medica, (1996) Vol. 62, No. 2, pp. 122-125.
ISSN: 0032-0943.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The antimicrobial mechanism of totarol was studied using Pseudomonas aeruginosa IFO 3080. This diterpene inhibited oxygen consumption and respiratory-driven proton translocation in whole cells, and oxidation of NADH in membrane preparation. NADH-**cytochrome c** reductase was inhibited by totarol while **cytochrome c** oxidase was not. NADH-DPIP reductase and NADH-CoQ reductase were also inhibited. The site of respiratory inhibition of totarol was thought to be near CoQ in the bacterial electron transport chain.

=> d 6 ibib abs

L7 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1983:402840 CAPLUS
DOCUMENT NUMBER: 99:2840
TITLE: The oxidation of methylated amines by the methylotrophic bacterium Methylophilus methylotrophus
AUTHOR(S): Burton, S. M.; Byrom, D.; Carver, M.; Jones, G. D. D.; Jones, C. W.
CORPORATE SOURCE: Dep. Biochem., Univ. Leicester, Leicester, LE1 7RH, UK
SOURCE: FEMS Microbiol. Lett. (1983), 17(1-2-3), 185-90
CODEN: FMLED7; ISSN: 0378-1097
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Respiratory activity was studied in M. methylotrophus cells grown on trimethylamine with the addn. of dimethylamine, methylamine, methanol, and **ascorbate**-N,N,N',N'-tetramethyl-p-phenylenediamine. Whole cells of M. methylotrophus grown on trimethylamine contained b- and c-type cytochromes, together with cytochromes o and(or) Cco, as the major potential oxidase(s); a3 but not a, was also detected. Such cells exhibited a low rate of endogenous respiration which was dramatically stimulated by the addn. of the other substrates. The anal. of fractions prepd. from M. methylotrophus showed that virtually all of the methylamine dehydrogenase and methanol dehydrogenase activities, together with >1/2 of the **cytochrome c**, were present in the periplasm, whereas all of the assayable dimethylamine monooxygenase and .apprx.2/3 of the trimethylamine dehydrogenase activities were present in the cytoplasm. The membranes contained all of the **NADH oxidase** activity and the b-type cytochromes. Apparently, trimethylamine dehydrogenase is assocd. with the cytoplasmic side of the membrane and donates reducing equivs. to the respiratory chain at the level of **ubiquinone**/cytochrome b, whereas methylamine dehydrogenase is loosely attached to the periplasmic side of the membrane and probably interacts with **cytochrome c**; no amicyanin was detected.

=> d 7 ibib abs

L7 ANSWER 7 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1977:115822 BIOSIS
DOCUMENT NUMBER: BA63:10686
TITLE: MEMBRANE BOUND RESPIRATORY CHAIN OF SPIRILLUM-ITERSONII.
AUTHOR(S): DAILEY H A JR
SOURCE: J BACTERIOL, (1976) 127 (3), 1286-1291.
CODEN: JOBAAY. ISSN: 0021-9193.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable

AB The membrane-bound respiratory system of the gram-negative bacterium S. itersonii was investigated. It contains cytochromes b (558), c (550), and o (558) and .beta.-dihydro-NADH and succinate oxidase activities under all growth conditions. It produces D-lactate and .alpha.-glycerophosphate dehydrogenases when grown with lactate or glycerol as sole C source. Membrane-bound malate dehydrogenase was not detectable under any conditions, although there is high activity of soluble NADH: malate dehydrogenase. When grown with O2 as the sole terminal electron acceptor, .apprx. 60% of the total b-type cytochrome is present as cytochrome o, whereas only 40% is present as cytochrome o in cells grown with nitrate in the presence of O2. NADH and succinate oxidase are inhibited by azide, cyanide, antimycin A and 2-n-heptyl-4-hydroxyquinoline-N-oxide at low concentrations. The ability of these inhibitors to completely inhibit oxidase activity at low concentrations and their effects upon the aerobic steady-state reduction levels of b- and c-type cytochromes and the aerobic steady-state reduction levels obtained wiht NADH, succinate and **ascorbate**-dichlorophenolindophenol suggest the presence of an unbranched respiratory chain in S. itersonii with the order **ubiquinone** .fwdarw. b .fwdarw. c .fwdarw. o .fwdarw. O2.

=> d 8 ibib abs

L7 ANSWER 8 OF 9 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1977:189538 BIOSIS
DOCUMENT NUMBER: BA64:11902
TITLE: THE SYSTEMIC FUNGICIDE TRIDEMORPH AS A DUAL SITE INHIBITOR OF THE RESPIRATORY CHAIN OF ELECTRON TRANSFER PARTICLES FROM BEEF HEART MITOCHONDRIA.
AUTHOR(S): MUELLER W; SCHEWE T
SOURCE: ACTA BIOL MED GER, (1976) 35 (6), 693-708.
CODEN: ABMGAJ. ISSN: 0001-5318.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable

AB Tridemorph (N-tridecyl-2,6-dimethylmorpholine) inhibits both the **NADH-oxidase** and the succinate-**cytochrome c** oxidoreductase system of non-phosphorylating electron transfer particles from beef heart. The concentration required for half-inhibition was 3.4 .mu.M and 24 .mu.M, respectively. Two different sites of action in the respiratory chain could be localized using difference spectroscopy and measurements of enzymic activities in various partial systems. The inhibition of the NADH-**ubiquinone** oxidoreductase activity, the suppression of the NADH-induced reduction of all cytochromes and the insensitivity of the NADH-ferricyanide oxydoreductase system indicate a site of action similar to rotenone. The partial suppression of the succinate-induced reduction of cytochrome b with simultaneous complete inhibition of the reduction of the other cytochromes indicate an additional site of action analogous to antimycin A. Both inhibitory actions appeared instantaneously after the addition of tridemorph and were counteracted by serum albumin. Tridemorph inhibited the oxidation of

external ferrocytochrome c but not that of **ascorbate** /tetra-methyl-p-phenylene-diamine-HCl (TMPID) showing that it is not a true inhibitor ofthe cytochrome oxidase. The TMPD-induced bypass of the succinate oxidation was inhibited as well. The possible role of the inhibition of the main pathway of the respiratory chain for the fungicidal action of tridemorph is disucssed.

=> d 9 ibib abs

L7 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1972:561840 CAPLUS
DOCUMENT NUMBER: 77:161840
TITLE: Comparison of the **NADH oxidase** electron transport systems of two obligately chemolithotrophic bacteria
AUTHOR(S): Sadler, Martha H.; Johnson, Emmett J.
CORPORATE SOURCE: Sch. Md., Tulane Univ., New Orleans, La., USA
SOURCE: Biochim. Biophys. Acta (1972), 283(1), 167-79
CODEN: BBACAQ
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The **NADH oxidase** electron-transport systems of two obligate autotrophs were investigated. Cytochromes c547, c550, c552, b or c554, b558 and a were found in Thiobacillus neapolitanus. and cytochromes c549, 551, c or b555, a1, and a or a3 in T. thioparus, A sol. cytochrome c552 not present in the particulate fractions was detected in T. neapolitanus. Low potential c-type cytochromes were found in both organisms. NADH reduced both cytochromes c547 and c550 in the large particle fraction of T. neapolitanus, but only c550 in the small particle fraction. Both organisms contained the **ubiquinone**, Q-8. The levels of flavine, quinone, and **cytochrome c** were comparable to those of heterotrophic bacteria. No naphthoquinone was detected. The levels of NADH and **ascorbate** oxidases were similar to those of heterotrophic bacteria, while NADH dehydrogenase and **ascorbate**:N, N, N', N'-tetramethyl-p-phenylenediamine.2HCl oxidase levels were higher. In T. thioparus, **NADH oxidase** activity was located exclusively in the large-particle fraction, and in T. neapolitanus in both the large- and small-particle fractions. The **NADH oxidase** activities of both organisms were sensitive to inhibitors usually employed in studies of electron transport. **NADH oxidase** of T. thioparus was completely inhibited by KCN, while that of T. neapolitanus was never inhibited by more than 80. **Ascorbate** and **ascorbate**:TMPD oxidases were sensitive to KCN but insensitive to 2-heptyl-4-hydroxyquinoline N-oxide. Electron-transport pathways are proposed for both organisms.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	45.21	45.36

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.35	-2.35

STN INTERNATIONAL LOGOFF AT 13:01:49 ON 07 AUG 2001